IN THE UNITED STATES DISTRICT COURT FOR NORTHERN DISTRICT OF MISSISSIPPI, WESTERN DIVISION

FRED BECK, ET AL.,)
)
Plaintiff,)
)
vs.) No. 3:03CV60-P-D
)
KOPPERS, INC., ET AL.,)
)
Defendants.)
_____)

DEPOSITION OF JAMES DAHLGREN, M.D.
SANTA MONICA, CALIFORNIA
TUESDAY, APRIL 5, 2005
VOLUME 1

Reported by:
VIRGINIA PETERAITIS
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Deposition of JAMES DAHLGREN, M.D. Volume 1, taken on behalf of Defendants, at 2811 Wilshire Boulevard, Suite 510, Santa Monica, California, beginning at 9:00 a.m. and ending at 4:00 p.m. on Tuesday, April 5, 2005, before VIRGINIA PETERAITIS, Certified Shorthand Reporter No. 6205.

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INDEX

Santa Monica, California, Tuesday, April 5, 2005
9:00 a.m. - 4:00 p.m.

JAMES DAHLGREN, M.D.
having been first administered an oath, was examined and testified as follows:

EXAMINATION

BY MR. HOPP:

Q Good morning, Dr. Dahlgren. We met a few minutes ago and I'm Anthony Hopp and I represent several defendants in this case and I'm here for your expert deposition. I know you've done this many times before and likely as often as I have.

Let me state a few ground rules so we're clear. I'm going to be asking a series of questions and it's important that you answer out loud yes or no, as opposed to shakes or nods of the head. When appropriate answer yes or no or verbally, as opposed to uh-huh or huh-uh and that's for the court reporter's benefit so we have a clear record.

If you answer a question, I'll assume you understood it. If you don't hear a question, don't understand a question, don't like a question, please ask me to rephrase or restate it and I'll be happy to do that.

Does all that sound fair?

A Yes.

Q Do you have anywhere in your office a current up-to-date CV?

A Yes.

Q I'll ask at a break that we get a copy and mark that as an exhibit and dispense with some of the more tedious stuff that we have to go through.

Is that okay with you?

A Yes.

Q Do you currently practice medicine?

A Yes.

Q Where?

A Here in this office.

Q We are in Santa Monica, California?

A Correct.

Q Whom do you treat?

A I have a small treatment practice, treat a handful of patients at this point in time.

Q For what do you treat patients?

A For toxic poisoning of various kinds and that's my specialty.

Q How often do you see patients in this office for any purpose?

A Two to three days a week I see patients in the

office here.

Q You drew a distinction earlier between your treatment practice and some other sort of practice, do you distinguish between treatment and some other medicine in your practice?

A Yes, patients I see for evaluation and not for treatment.

Q You say you have a small treatment practice?

A Yes.

Q How small? Give me a ballpark.

A Probably 200 patients or so.

Q And the remainder of the patients you see are evaluations only?

A Correct.

Q In the last 12 months, how many people would you say you've evaluated?

A I don't know. I don't have that figure in my mind.

Q Hundreds, dozens, thousands?

A Hundreds probably.

Q How do you normally get connected with patients for the purpose of evaluation? Do people call to come see you or some are brought to you by others?

A They usually call to make appointments.

Q What I'm driving at -- it was a poor question.

Are most of the patients you see for evaluation referred by lawyers?

A Some are, not all.

Q What other sources are there for the patients you see for evaluation?

A Well, insurance company adjusters, other doctors, patients, companies, unions, a whole variety of different sources.

Q What sorts of evaluations do you do when you evaluate patients?

A I'm an internist, and I do what would be called internal medicine evaluations and take a history from the patient and examine them and collect any medical records, perform lab tests; the usual approach to patients.

Q We're here today in a lawsuit dealing with an alleged toxic exposure. Are most of the people you see for evaluation people who claim some kind of toxic exposure?

A Yes. Most patients I see have been exposed to some kind of toxins and that's my sub-specialty.

Q That's why they come to you, they claim to be exposed and you're evaluating them for that purpose; is that right?

A Yes.

Q Let's talk about your treatment practice. You

said 200 or so patients you continue to treat. What do you treat them for? What conditions are you treating?
A Well, lead poisoning, pesticide poisoning, mold poisoning, a variety of things.
Q Are you currently treating a group of firemen in New York City?
A Yes.
Q What for?
A What's called WTC syndrome.
Q What is WTC syndrome?
A World Trade Center syndrome.
Q Describe briefly what's involved in the World Trade Center syndrome.
A Following the World Trade Center collapse, there were several thousand rescue workers, including firemen, who were exposed to the smoke, dust and fumes from the Trade Center collapse, causing many of them, if not all, to become ill to one degree or another.
The precise ticket or chemicals involved has not really been identified. We have been using PCBs and dioxins as markers of exposure and those two chemical classes do constitute at least one element of why they got sick, but many, many other chemicals, heavy metals, fine particles, dust, many organic chemicals that were also inhaled or absorbed during the episode contribute to

the illnesses that are seen in these individuals.
Q Is there a typical presentation for the illness or are they all sick in the same way?
A They're sick in similar ways and it's characterized by chronic cough, shortness of breath and varying degrees of respiratory complaints and can have an asthma-like picture, and some have a more interstitial lung problem, so there is some variety but most all of them have respiratory involvement.
Second area is the neurologic system. Most of them complained of headaches, light-headedness, memory disturbance, altered mood, depression, anxiety, bad dreams, and a whole spectrum of neurologic complaints, which is compatible with the mixture of chemicals that we saw there.
And then a fairly significant number have skin, gastrointestinal immune system problems, probably related to the chemicals they were exposed to, as well, and thus decreases reproductive function, decreased libido, decreased energy, chronic fatigue and all these things are somewhat variable.
Two areas that are quite consistent are the respiratory and neurologic, which affected almost all the people at the Trade Center during the days and weeks after the collapse of the two buildings.

Q Have you taken blood samples from these patients for the purpose of evaluation of the dioxin and furan levels in their blood?
A PCB levels, yes.
Q Have you reported those in some sort of publication or articles you've written on that?
A Yes, I presented the data on the New York City firemen that we studied in Berlin at the dioxin 2004 meeting, and the four-page abstract, which was a short article was published as part of the proceedings from that meeting and was published on the Internet and by way of CD ROM.
We're preparing an expanded paper for publication in Hemisphere, a journal that publishes articles arising out of the dioxin meeting.
Q You spoke earlier of your treating these patients. What's the goal of your treatment for these individuals from the World Trade Center?
A To reduce their body burden of the PCBs and dioxins is one aspect, but the real purpose is to make them feel better and reduce their symptoms or eliminate their symptoms and get them back to a more normal state.
Q Is there a particular method you're using to try to reduce their body burden of PCBs and dioxins?
A Yes.

Q Describe that.
A It's a protocol that involves the administration of Niacin or vitamin B 3, I believe it is, and exercise for 30 minutes on a treadmill or bicycle, followed 3 to 5 hours of sauna, increasing the excretion of the chemicals out through the skin and, as part of that, we give vegetable oil and replenish the various minerals lost from sweating, and the mineral oils and vegetable oils are intended to promote the excretion of the PCBs and dioxins through the stool.
Q Is there a label or a name that this treatment regime has?
A Just a detoxification treatment.
Q Have you heard it called the Hubbard treatment or the Hubbard regime?
A I've heard that.
Q Who is that named after?
A L. Ron Hubbard, founder of Scientology.
Q Did Mr. Hubbard develop this treatment regime or was it named after him for some other reason, if you know?
A I believe it's named after him because he was the first guy who wrote up the technique and published it.
The utilization of sauna therapy, which is

actually an old therapy, has been used by most cultures as a therapeutic modality for thousands of years, so I don't think he invented it but described the utilization with the Niacin as an adjunct to it.

Q So somewhere we can find an article written by Mr. Hubbard which describes the treatment regime with sauna and Niacin?

A There's a number of papers published over the years on this detoxification method. It was used in the early 80's to treat patients in northern Michigan exposed to polybrominated biphenyls and was published by a Dr. Schnare, I believe is the name.

There was another paper published in the late 80's from Yugoslavia on patients who worked in a PCB factory. I participated with Dr. Tretjak, who was from Yugoslavia, the patient's doctor, and was brought to Los Angeles, and she was put through the treatment program that I just told you about and that patient was published in a case report by Dr. Tretjak.

Q That was a single Yugoslavian patient that Dr. Tretjak brought to the United States for treatment?

A Yes.

Q And did Dr. Tretjak publish the paper?

A Yes.

Q Were you a co-author?

A No, I was not. But we showed in the paper there was a significant reduction in PCBs -- we measured them before and after, and measured the skin oil PCB levels during the treatment, and my main role was as a lab person, establishing the protocols for the laboratory.

Q Let's go back to the fire fighters. I know you say it was the Hubbard method originally written by Mr. Hubbard.

Is the current protocol you're using taken directly from Mr. Hubbard's paper or have you modified it in some way?

A We made some modifications.

Q How have you modified the Hubbard method?

A I don't call it the Hubbard method but a detoxification method.

Q How is your detoxification method different from the Hubbard method as written by him?

A We substituted or added cholestyramine to the regimen to reduce the PCBs. We changed the protocol and made a modification because people have a hard time spending 5 hours in the sauna, and we do it for 2 or 3 hours and find we're getting good results from using a shorter time frame.

Q Is your protocol for the World Trade Center

rescue workers written up anywhere?

A It's been written up in the paper briefly, referring to those earlier papers which describe it in more detail.

Q I want to make sure I understand. There was a paper published by Mr. Hubbard, and the abstract you recently published. Has someone else other than you or Mr. Hubbard written some sort of intermediate paper that describes further modifications to the Hubbard method and why they would be effective and what the reasons for the modifications are?

A I told you about two other papers written by Dr. Schnare and Dr. Tretjak and they didn't change the original protocol.

Q With respect to the fire fighters or let's call them the World Trade Center cohorts, you took measurements of dioxin and furan and PCB levels in their blood before starting the regimen?

A Yes.

Q Have you measured the dioxins and furans and PCBs in their blood during the treatment?

A No, afterward.

Q And is the treatment completed?

A Yes.

Q And you took measurements subsequent to the

treatment; correct?

A Yes.

Q And how, if at all, did the dioxin, furan and PCB levels in the blood change?

A It went down by a significant percentage -- different effects. The most important effect was the TEQs dropped by 60 percent in the 7 patients we measured. The largest reductions were in the patients with the highest levels but there was a consistent reduction of TEQs.

Q You measured 7 patients?

A Yes.

Q Were there more than 7 patients that went through the treatment program?

A Yes, several hundred have gone through the treatment program in New York City.

Q Why did you select 7 to measure?

A Economic constraints. It's very expensive to do the testing.

Q How did you pick the 7 out of the hundred --

A They were the first people that were available as soon as the money was made available.

Q So, I'm sorry, I didn't understand that. The first people who were made available after the money was made available?

A Well, they were treating patients on a regular basis in the clinic in New York and then found the money to pay for the testing and they donated the money from various fund-raising activities carried out by the clinic. We just did the next 7 patients that came through.

Q You did the 7 patients. Were those 7 patients that you did both before and after, did you follow the 7 patients for the purpose of measuring the effect of this treatment technique?

A Yes.

Q So the first 7 who came through, they were the ones who were selected to be tested before and after?

A Correct.

Q Do you believe those 7 are representative of the entire cohort of World Trade Center rescue workers?

A Yes.

Q Why?

A I don't have any reason to think they were any different than anybody else who had similar histories.

Q Similar histories in terms of the exposure, that is the work they did at the WTC?

A Yes, and in terms of the symptoms and health findings.

Q Now, with respect to the 7 you treated, you had

no way of knowing what their dioxin, furan and PCB levels were before you first encountered them?

A Right.

Q And you first encountered them after the World Trade Center clean-up; is that correct?

A Yes.

Q If one of the 7 had a higher level before the WTC clean-up, your method would not have picked that up?

A That's correct.

Q Are dioxins hydrophilic or hydrophobic?

A Hydrophobic.

Q So they have preference for fat cells?

A Yes.

Q Is that part of the reason for treatment, to pull the dioxin out of the fat cells?

A Well, the excretion patterns for dioxins and PCBs and furans is primarily through the bile and they don't -- because they're not water soluble, they don't excrete in the urine to any appreciable degree.

And the problem is they're secreted along with bile salts which then are reabsorbed in the small intestine, so PCBs and dioxins undergo what is called an enteropatic circulation and they're secreted in the bile and reabsorbed so that they stay in the body, and that's why the half life for dioxins and PCBs is measured in

years -- 5, 6, 10 years duration, depending on which particular congener we're talking about.

Q I think I understood your answer but have trouble relating it back to my question.

How does your treatment regimen help the excretion of PCBs, dioxins and furans from the WTC rescue workers?

A Well, by using the skin as an excretory organ. The skin surface area is large and by putting the patient in the sauna, after they have exercised and taken Niacin, there is a very large increase in the amount of skin oil and sweat which is produced and, therefore, you get a net decrease in the amount of PCB that stays in the body. Secondary it blocks the reabsorption of the bile salts in the small intestine and promotes the excretion of the chemicals through that route, as well, and that's what I was saying about the cholestyramine as a more effective way of accomplishing that blockage.

Q So the Niacin helps -- again I'm trying to understand the theory of the mechanism. Dioxin is stored in fat?

A Yes.

Q Or fat cells of the body?

A Yes.

Q Because it is hydrophobic it doesn't like the

water, it doesn't like the blood?

A It goes in the blood but goes in the -- tends to be absorbed onto the fat in the blood. Hydrophobic simply means it's not water-soluble and doesn't lend itself to excretory excretion in the kidney.

Q So the Niacin increases the blood flow to the skin and increases the enzyme activity in various cells of the body to promote turnover of the lipid solubles, and Niacin is used to help reduce cholesterol levels and speeds up the breakdown of cholesterol.

So the Niacin helps blood flow to the skin and helps the dioxin to pass through the skin barrier and then you excrete it actually to the surface of your skin through the sweat?

A Skin oils of the skin are increased when you increase the sweating and increase the body temperature and a greater percentage of the body burden shows up on the skin as a result of this technique.

Q Has there been any effort to measure how many dioxins come through in the sweat?

A I said we measured PCBs in the skin oil of the patients in Yugoslavia and did not do dioxins but did PCBs as a marker. A lot of these chemicals behave the same way and you can't measure all of them, and we use PCBs as a marker, and the concentration in the skin oil

was high, much higher than normal, indicating that was indeed an excretory pathway for this type of chemical.
Q That was the one patient from Yugoslavia?
A Yes.
Q Has there been any effort in the Trade Center workers to measure what comes from their skin oil?
A No. We have no money for that.
Q I think you said something about the small intestine and the reuptake of dioxin and then with cholestyramine. So the feces is another avenue of getting rid of dioxins in this technique?
A Yes. That's the techniques used.
Q Has there been any evidence in the World Trade Center cohort to measure dioxin in stool samples?
A No.
Q Now, we talked about Dr. Tretjak and, again, you'll have to help me and I apologize for asking the question again.
Dr. Tretjak was the doctor who brought the Yugoslavian patient here to L.A.?
A Yes.
Q And then you administered this techniques or this therapy to the WTC workers.
Other than the two events, are there any other documented cases of this detoxification technique being

administered?
A I told you about the techniques used to treat the patients with polybrominated biphenyl exposure in the early 80's published by Dr. Schnare.
Q So Schnare and Tretjak are the most recent ones; right?
A Yes.
Q Any others?
A I think there is a publication by a Russian author where they used a technique to treat patients of Chernobyl, and I don't recall the author's name, and I think there are some other papers that I have to go look at them and didn't have personal involvement with them so I don't recall the details.
Q We all know what happened at Chernobyl. Was the contaminant concern for the purpose of this treatment PCBs, dioxins and furans or what is the radioactive --
A It was the radioactive components.
Q I want to go back to more generally your practice. How many patients did you see in March of 2005, if you know?
A This month?
Q This last full month.
A I probably saw maybe 50, 60 patients.
Q And how many of those again roughly were

evaluation and treatment?
A 5 or 6 for treatment and the rest for evaluation.
Q If say, for example, I wanted to make an appointment with you for an examination, can I do that? Would you accept me as a patient?
A I don't do general internal medicine any longer so depending what your problem was.
Q And irrespective of the fact that I'm a lawyer on the case, if someone called to make an appointment with you, would they need to present with some sort of claimed toxic injury before you'd see them?
A No, but it would be patients who have problems with a toxicity issue and not necessarily a claim for a toxic injury.
Q So you don't take patients who just call you up and you need to sort of pre-qualify them?
A I'm not in internal medicine practice any longer, and I did that for many years and not lately.
Q Do you take Blue Cross or Blue Shield?
A Well, I will help the patient bill their insurance but I don't accept the assignment or don't belong to a PPO.
Q Do you treat Medicare or Medic-aid patients?
A Yes. Again I don't accept assignment.

Q You said you're not a member of any PPOs. Are you a member of any HMOs?
A No.
Q Do you take any type of insurance?
A Yes, like I said, but we don't accept -- in other words, accepting the assignment means you accept whatever the insurance company decides to pay you and we don't do that.
Q You'll take reimbursement from insurance but you will bill at your rates, as opposed to insurance rates?
A That's correct.
Q Would you describe for me your relationship with UCLA?
A I'm on the clinical faculty and have been there many years.
Q You're paid by UCLA?
A No.
Q Do you have an office at UCLA?
A No.
Q Do you teach classes at UCLA right now?
A I don't teach classes. I'm on the clinical faculty and attend rounds and meet with the residents and students periodically to talk about cases, but I don't give lectures or run a class. I've done some of that in

the past but not lately.
Q When was the last time you ran a class or gave lectures at UCLA?
A Probably 3, 4 years ago.
Q Was it a class or lecture?
A The last major teaching activity that I had was doctoring classes, which met once a week for three hours with the students, and that was the last time and that was probably several years ago the last time I did doctoring, and it's very time-consuming and difficult to find the time in the week to do it.
Q What's doctoring?
A It's a class they have in UCLA, teaching students a little bit about the broader aspects of taking care of patients.
Q And you said it was several years ago. Was it in the 90's? 80's?
A 90's, between 1995 and 2000 was the doctoring class.
Q Did you teach the doctoring class for one semester, two semesters --
A Five years.
Q Was it the same group of students for five years or get a new group every year?
A Different students. There is doctoring 1, 2

and 3 and depends on which one I was involved in.
Q I hate to make you repeat it but you said once a week for three hours?
A Yes, for a semester.
Q And that went on for five years?
A Yes.
Q And were you paid for that?
A No.
Q It was voluntary work?
A All of my clinical faculty work is voluntary, that's correct.
Q Other than the doctoring class, have you taught any other classes at UCLA in the last ten years?
A Yes.
Q Can you tell me what they were?
A I've given lectures on toxicity issues to the students and public health classes, residents in occupational medicine and occupational environmental medicine.
We used to have an elective where students would come and spend time here in the office from anywhere from four to six weeks, and I've given up on that and it's too time-consuming. Those are the main things I've done, tending round on a regular basis.
Q What's the last time you tended rounds?

A A few months ago.
Q How does this occur? Do you have a contact at UCLA that calls you and says to come and tend rounds?
A They send a schedule and e-mail these days.
Q So you're on some list of doctors who are available to come and tend rounds, if they need it?
A Yes.
Q Are you still on that list?
A Yes.
Q Do you have a contact at UCLA School of Medicine where if we wanted to contact them to get information about your involvement, is there a point person to talk to?
A Phil Harbor.
Q H-a-r-b-o-r?
A Yes.
Q What's his position with UCLA?
A He's the paid faculty member they have there.
Q Paid faculty member in what?
A Occupational environmental medicine.
Q If I were to call UCLA and try to get you there, do you have a voice mail account or would they take a message for you?
A No, I don't have a message sender there.
Q Is it accurate to say that you are an assistant

professor of clinical medicine at UCLA?
A Clinical assistant professor they call it.
Q And is there a deference between assistant professor of clinical medicine and a clinical assistant professor?
A No, just word order.
Q Has anyone complained to you of your use of UCLA's name on your published papers?
A No, you're supposed to put UCLA's name on the published papers.
Q Has anyone at UCLA ever asked you to stop representing yourself as being associated with UCLA?
A No.
Q Are you involved in any litigation or claims regarding your use of UCLA's name on your published works?
A No.
Q Are you board certified?
A Yes.
Q In what?
A Internal medicine.
Q Any others?
A No.
Q Are you a pediatrician?
A No.

Q Have you ever practiced pediatric medicine?
A No.
Q Are you an oncologist?
A No.
Q Is it possible to be board certified as a toxicologist?
A Yes.
Q Have you ever been board certified as a toxicologist?
A No.
Q How does one become board certified as a toxicologist?
A Join one of the groups that has a board certification process and go through their procedures.
Q Is there some particular reason you've not done that?
A I've not attended the meetings, the Society of Toxicology or American College of Toxicology and am not active in either organization and never belonged to them and never -- I've got enough professional meetings to go to in the American College of Occupational Environmental Medicine and other meetings I attend, as the dioxin meetings, adding another group of meetings, which is something I've not done to now.
Q Is attending the regular meetings of a group

like that one of the conditions of maintaining your board certification?
A I don't know.
Q In any event, if you were to become board certified in the field of toxicology, they'd invite you to meetings?
A It's the other way around, you go to meetings and I think -- I have not checked it out, but I believe that you apply to take the exam they give.
Q And there is an examination you have to pass in order to obtain board certification?
A Yes, that's my understanding. That's how they do it in the other disciplines, they administer a test to make certain you have a certain body of knowledge.
Q Are you familiar with an organization called the Collegium Ramazinni?
A Yes.
Q Are you a member of it?
A No.
Q What is it?
A It's a group that Selikoff started years ago and it's where researchers in the field of occupational medicine get together and present papers and just another organization that exists.
Q Is there any particular reason you have not

joined the Collegium Ramazinni?
A No.
Q Forgive me if we've covered it but do you have a regular practice in medicine that deals with things other than occupational or chemical exposures?
A Environmental exposures.
Q So we can divide your patient load among environmental exposures, occupational and other general chemical exposures?
A Yes.
Q And those three exposure groups pretty much describe the entirety of your practice; is that correct?
A Yes.
Q When you see someone for evaluation, as opposed to treatment, do you typically make a diagnosis at the end of the diagnosis?
A You make a tentative diagnosis based on the history and physical examination. Sometimes you can't be certain but usually there is an impression that we have, a tentative diagnosis.
Q Now, when you reach a tentative diagnosis, do you typically send patients to other specialists for more in-depth treatment?
A In depth evaluation, you mean?
Q Sure, in-depth evaluation or treatment.

A We don't send patients typically to other specialists for treatment. Depends what it is. If patients have an acute appendix, we send him to a surgeon to have the appendix removed or a freshly diagnosed cancer that's not treated, I try to help them find an oncologist to treat their cancer.
Usually that is not the case and usually you're talking about some patients for evaluation and treatment who have come to us because of the issues of toxicity. We may even send them to other doctors for certain testing, pulmonary function studies or smell testing, neuropsychological testing or some other evaluations that would be done by other specialists but usually not for treatment, as a rule in my practice.
Q So other than in the instances you identified, typically do you see a patient, provide an evaluation report and that's the end of your involvement with the patient?
A No. Usually there is some follow-up testing done and possibly the communication of that information of the patient. It depends on the circumstances. There is no set rules for how we do it. It varies according to the patient and the problems.
Q Would you say more often than not you're involved in the follow-up evaluations or more often than

not your involvement ends with a single evaluation and issuance of a report?
A If it's an evaluation in the field, where we see a patient as part of an examination for, say, a group of patients, we probably would not see those patients again. If a patient comes to the office for an evaluation, we frequently see them again.
Q And who decides whether someone comes to the office or are seen in the field?
A If you're seeing a large number of people, it's much more practical to go to the field and see them rather than have them come to the office.
Q So smaller groups or individuals come to your office here in California?
A Correct.
Q For larger groups involved in say big claims or lawsuits, you go, in this instance, to Mississippi?
A Yes.
Q And you see the Mississippi patients in this case and evaluate them and provide an evaluation report and that's the end of your involvement, other than expert testimony?
A That's correct.
Q There was a lawsuit I believe that was recently settled involving the town of Jerome in Florida?

A Yes.
Q Are you familiar with that lawsuit?
A Yes.
Q Were you involved in evaluating people in Jerome for --
A Yes.
Q And I heard that lawsuit is settled. Has anybody communicated that to you?
A I heard that, as well.
Q Are you involved in any follow-up treatment or care for the Jerome, Florida residents that you evaluated in that case?
A No.
Q And what was the constituent of concern or contaminant of concern in that case?
A There was creosote contamination of the ground and water supply people were using.
Q As a physician, have you treated someone for pancreatic cancer?
A Yes.
Q How often?
A Gosh, many times. Pancreatic cancer was -- I guess probably in the range of 40, 50 patients.
Q When did that occur?
A Back in primary care and residency in medical

school and training, really part of my career. In recent years I'm not treating patients for cancer and I refer them to oncologists.
Q I want to get a better handle -- I have a list of conditions and I imagine the answers will be similar.
In the early part of your career -- and I know we asked for your CV and we'll get all this, but when did you graduate medical school, what year?
A 1968.
Q And during what years were you involved in the primary care for patients?
A Until 1993.
Q So when we talked about pancreatic cancer, you said you treated patients as a primary care physician and a resident in medical school, et cetera.
Was that between the late 1960's and 1993?
A Yes.
Q Have you ever treated anyone for stomach cancer?
A Yes.
Q How many people?
A In the range of 15, 20.
Q During what period did you do that?
A Same period we talked about, '68 to '93.
Q Have you ever treated anybody for breast

cancer?
A Yes.
Q How many?
A Probably a hundred.
Q During what period?
A Same period.
Q Have you ever treated anyone for diabetes?
A Yes.
Q How many?
A I would say a couple of thousand.
Q During what period?
A Same period.
Q Did you ever treat anybody for liver conditions, including elevated liver enzymes?
A Yes.
Q How many?
A Hundreds.
Q During what period?
A Again, the same time frame, '68 to '93.
Q Did you ever treat anybody for sclerosis of the liver?
A Yes.
Q How many?
A Dozens of patients.
Q During what period?

A Same period.
Q Have you ever treated anyone for any form of skin cancer?
A Yes.
Q How many?
A Again, I don't have a precise number but it's a common condition.
Q Same time period?
A Yes.
Q Have you ever treated anyone for cardiovascular disease?
A Yes.
Q How many?
A Thousands. It's the most common disease that internists treat.
Q During what period?
A '68 to '93.
Q Have you ever treated anyone for asthma?
A Yes.
Q How many?
A I would say thousands of patients.
Q During what period?
A Same time period.
Q Have you ever treated anyone for chronic bronchitis?

A That's any kind of an adverse problem arising in utero, around the time of birth, and, you know, obviously the patients who have birth defects or are premature or low birth weight or have high membrane disease or infant respiratory distress syndrome or other complications around the birth process, they would be taken care of by doctors who specialize in that area and that's not my area of specialty.
Q That's something a neonatologist might take care of?
A Yes.
Q Would your answer be the same for low birth weight, low birth length, low birth head size, asymmetrical growth retardation or reduced mental and motor development.
A Yes.
Q These are all things that a neonatologist would take care of?
A Correct.
Q One of the patients in this case has diabetes. Are you aware of that?
A Yes.
Q Did you send her to a specialist who treats diabetics?
A No, I didn't refer her to anyone and didn't

A Yes.
Q How many?
A I'd say hundreds to thousands.
Q Same time frame?
A Yes.
Q Have you ever treated anyone for obstructive lung disease?
A Yes.
Q How many?
A Thousands.
Q During the same time period?
A Yes.
Q Have you ever treated anyone for dental problems?
A No, I don't treat dental problems.
Q Have you ever treated anyone for birth effects, including small birth size?
A No, I've not treated anybody for small birth size.
Q Birth effects is a term I've taken from your report and I want to make it clear I'm not talking about birth defects.
Is there a particular doctor who treats patients for birth effects, like the ones identified in your report in this case?

participate in her care at all.
Q One of the plaintiffs in this case has elevated liver enzymes, are you aware of that?
A Yes.
Q Did you refer that patient to a specialist in liver disease?
A No, I didn't refer any of these patients to any doctors for any treatment for any condition at any time.
Q And I want to go through the list. Same goes for cardiovascular disease, asthma, chronic bronchitis, obstructive lung disease, dental problems --
A I have never referred any of these patients at any time for any treatment for any of their conditions.
Q Is it your opinion that any of the plaintiffs in this case have an increased risk of developing cancer later in their life?
A Yes.
Q Who?
A All of them.
Q Do you know what types of cancer the plaintiffs are at risk for developing later in life?
A All types of cancer. It depends on which organ responds to the cancer-causing agents. These particular cancer-causing agents arising from the Koppers facility -- dioxins, PAHs, pentachlorophenols -- are carcinogenic

and reach almost all the tissues associated with numerous types of cancers, and I would say that any type of cancer has an increased risk for all types of cancer.

Q Let's explore that. Are you aware of any substance which is a canned carcinogen that causes cancer in every organ?

A Yes.

Q What is that?

A PAHs and the dioxins that I identified and I'd say that chromium also fits in that category, as does asbestos and numerous other carcinogens. Whatever tissue they reach, they increase the risk of cancer in that organ.

The organs with the highest exposure tend to be where you see the most cancers occur and the organs with the most rapid turnover and the highest exposure also tend to be organs where you see the increase of the number of cancers in the specific organ.

Dioxin, for example, is the most potent carcinogen that we know of and causes cancer in the lowest doses of any chemical and distributes widely throughout. Wherever it's looked for, it's basically been found, and certain tissue with higher lipid content, such as adipose tissue, brain, liver have higher concentrations, but it's basically distributed throughout

the body, as far as I know, to everywhere.

Q We'll spend quite a bit of time and probably not today going through studies you've cited and talking about the epidemiology of the various agents, but I want to understand your general approach here, and let's take asbestos. It's closely tied to lunch cancer; is that right?

A If you take studies of asbestos workers, the leading cause of death for asbestos exposed workers who smoke cigarettes is lung cancer, yes.

Q Are you aware of any epidemiologic literature that says asbestos can cause increased risk of kidney cancer?

A Yes.

Q Are you aware of any -- I'm trying to pick one that's not in the literature and test the limits of your theory.

Are you aware of any epidemiologic literature that says asbestos is associated with increased risk of liver cancer?

MR. LUNDY: I will object to the form of the question insofar as it says the limits of his theory, when he's sitting here giving you epidemiological evidence and not necessarily his theory.

MR. HOPP: I understand the objection --

MR. LUNDY: You can answer the question.

BY MR. HOPP:

Q Is asbestos associated with liver cancer in the epidemiological literature?

A I don't remember for sure.

Q Is it your belief or opinion that because asbestos is a potent carcinogen that where it is lodged in the liver can cause cancer in the liver?

A I think that's true, yes.

Q And so a potent carcinogen like dioxin, PAH, asbestos or chromium can cause cancer in any organ they reach; is that right?

A Yes.

Q The only way they couldn't increase the risk of cancer in a particular organ is that somehow the body doesn't distribute that particular substance to the organ?

A That's correct.

Q And since blood flows throughout the body, there are not many organs that don't have the potential at least for being a receptor for one of these contaminants; is that correct?

A That's correct.

Q Going back to the 7 living plaintiffs in this case, have you communicated to them that there is an

increased risk at cancer for all of their organs?

A No.

Q Have you recommended either to them personally or through their lawyers the follow-up care they should have in order to address this increased risk of cancer?

A Yes. As I said in my report, they all need regular medical monitoring.

Q And the medical monitoring protocol set forth in your report is something designed to address this increased risk of cancer?

A Yes.

Q Is there any particular reason you didn't tell the plaintiffs they were at an increased risk of cancer in all their organs?

A No, I don't have a reason that I didn't or that is particularly standing out in my mind. It's just not something I ordinarily do.

Q Why not?

A I don't have a particular reason why I don't sit down and go over that with them, because probably it's an unpleasant topic, and I just don't really feel the need to go there.

Q In this group of 7 living plaintiffs, are there any you identified who are at greater risk than the others for developing some particular form of cancer?

A No, I have not looked at the issue that way where I looked at the people with the highest level of exposure and the highest risk and the most other risk factors -- I have not looked at the data that way.
Q So your opinion is generally they're at a higher risk for all cancers, but you've not differentiated among plaintiffs or among cancers?
A Correct.
Q Are you familiar with the field of epidemiology?
A Yes.
Q Can you define epidemiology for us?
A Study of epidemics.
Q Any other definitions -- accepted definitions or is that it?
A I don't know what else to tell you. That's what the word means.
Q Are you prepared to offer opinions in this case in the field of epidemiology?
A Yes.
Q What makes you qualified to do that?
A Well, my background, training and experience. I've had extensive experience, published articles and took a course in epidemiology in the school of public health and it's been an integral part of my career in the

A I have some of the journals I subscribe to on a regular basis.
Q When we talk about the peer reviewers, I know you said that the editors send it out to two peer reviewers. Let's take an example so we're clear.
If you were to do a study that you wanted to publish in a peer reviewed literature and it had to do with heart issues, cardiovascular issues, is there a particular journal you'd send that to?
A Well, if it's about the treatment of heart disease with medication, you'd probably send it to Journal of Cardiology. If it's about cardiac disease arising from occupational exposure, you send it to Occupational Medicine Journal.
Q Fair enough. Each of the journals has someone whose job it is to take submissions and to decide whether or not to submit them for peer review; is that right?
A Yes.
Q There is an editor or series of editors for these peer reviewed journals?
A Yes.
Q Does the editor do some sort of triage and look at the article to see if it's worthy enough for peer review?
A That occasionally happens where the editor

last 30 years.
Q Let's talk about that. You rely on published sources as authority for your reports and opinions?
A Yes.
Q Much of what you rely on has been peer reviewed?
A Yes.
Q What does it mean for a study to be peer reviewed?
A You submit an abstract and a paper, draft of a paper, with your information in it, and the journal will then send that manuscript to reviewers, usually to two reviewers, and they will review the paper and decide whether it's got scientific merit and whether it's useful to the community that that particular journal is addressed to and, if they feel it's appropriate and scientific valid, they recommend it be published. If it's not recommended, it's not published. So that's what is meant by peer reviewed.
Q There are a series of peer reviewed journals that are available for doctors and epidemiologists or members of the general public; correct?
A Yes.
Q And you probably have a bunch here in your office; right?

makes the decision to not send it to a reviewer, if that's the question.
Q Does each journal have a list of reviewers that they then send papers out to for peer review?
A Yes.
Q And are there reviewers selected based on the subject of the paper, that is if it's occupational exposure to asbestos, are there reviewers who specialize in that discipline and review the paper?
A Usually you pick a reviewer that has some background, training and experience in the field so they can understand it and judge it appropriately.
Q Are you a peer reviewer for any journal?
A I have been.
Q Are you currently a peer reviewer?
A Yes.
Q What journals?
A Environmental Research, Journal of Occupational Environmental Medicine. Those are the two that come to mind at the moment and there are a couple of others that send me articles to review.
Q So Environmental Research and the second one is Journal of Occupational --
A And Environmental Medicine.
Q Now, what does a reviewer look for when he or

she receives a paper?
A Well, they look for the scientific validity and soundness of the study.
Q For example, you submitted papers to journals for peer review and non-peer review; is that right?
A I've never submitted anything to a non-peer review journal.
Q You've submitted papers to peer review journals?
A Yes.
Q Is it the reviewer's job to go back and double-check your data and see if you collected the data in the right way?
A As part of their assessment, they will look at the question of data and how the data was collected and how the sample was collected.
Q Isn't it true that the reviewers are looking more at the methodology and the documentation for the methodology and how the study was done, as opposed to getting the right results or that your numbers are correct?
A They also look at the numbers and that the statistics are done properly and the accounting is consistent, and they look at all aspects of the paper.
Q And then the reviewers will submit comments?

A Yes.
Q Usually in writing?
A Always in writing.
Q And the journal keeps those comments presumably?
A They usually forward them to the author so the author has the benefit. Frequently reviewers will ask for clarifications or changes or additions or subtractions even from the paper to make the paper more useful, let's say, to the community that it's prepared for.
Q Is it sometimes the case that the author will look at the reviewer's comments and make changes to the paper?
A Yes.
Q And then resubmit it?
A Yes.
Q Is it sometimes the case that the reviewers say that this paper should not be published and there is no second chance to submit it?
A That's correct.
MR. HOPP: We've gone for an hour, and should we take a comfort break?
MR. LUNDY: Sure.
(Recess.)

BY MR. HOPP:
Q Just to return to the New York City fire fighters study that we talked about earlier, at any point did you collect data on New York City fire fighters that did not respond to the World Trade Center cleanup to obtain a baseline number?
A No, we didn't. Dave Prezant the medical director for the fire department did do that, a PCB analysis on several hundred of the firemen and their values were a little higher than the background levels for the general population and I didn't see the data.
They had quite a few and, again, I don't know the exact number but in the range of 50 to 100 whose PCB levels using the Webb McCall technique were over 12, which was Dr. Prezant's cutoff, what he considered to be high. But the Webb McCall technique really is not the best technique to use but it's a quick and dirty way of getting PCB estimates and a relative concentration of PCBs from one person to another.
So they did have quite a few people that were high on the PCB count, and I think it's fair to say that the main thinking was that 9/11 was the reason for that.
Q I want to follow up on the prior answer. The name is Dave Prezant?
A Yes.

Q And he's the medical director for the New York City Fire Department?
A He and Dr. Kelly are two medical directors for the New York City Fire Department.
Q I want to understand. We asked about background, and you said he had collected data and he had some that were quite high with the levels of over 12, and that 9/11 was the reason for that.
The ones that were high, were they people who responded to the 9/11 in the cleanup?
A Yes.
Q But I want to go back to my earlier question. Did Dr. Prezant or anyone else to your knowledge collect data on New York City fire fighters who did not participate in the 9/11 cleanup, if any?
A Not to my knowledge.
Q Now, we were talking about peer review and published data. Is it generally accepted among people in your profession that a paper that's peer reviewed is in some way more reliable than one that has not been?
A That is a very complex question, and each paper I think has to be judged by some merit, and there are papers in the peer-reviewed literature that have serious faults with them and, vice versa, there are paper not peer-reviewed that are perfectly wonderful.

The idea of peer review is probably overly emphasized, and I understand why it's done, but I don't think you can make a sweeping statement that peer-reviewed stuff is good and non-peer-reviewed stuff is bad or anything like that.

Q Do you accept peer review as an indication of reliability?

A Well, it is certainly a process that is attempting to increase the reliability. As I say, there are papers published that are not very good, and you can say that the peer reviewers do a very good job, and I don't have any other -- I don't think just because it's peer-reviewed means it's good and, because it isn't, it's bad. It's part of the process and one has to judge the data based on all of the factors in the equation.

Q Let's talk about data. You rely on data from published sources; is that correct? For the purpose of your opinions in this case, you look at data contained in published papers?

A I have relied upon an extensive body of published information about the toxicity of the chemicals in this case.

Q And you depend on the data to be accurate?

A Yes, I do and, in a broad sweep, I think it's accurate, yes.

Q And if it's not accurate, if the data in the published papers that you rely on is not accurate, that can affect your conclusion; is that right?

The conclusions you draw from that data may be wrong?

A Anything is possible. There could be errors.

Q And when you take data from a published source, in order to put it into one of your reports or for other reasons, it's important you reproduce it accurately; is that correct?

A Yes. You should be faithful to what the article has stated.

Q And that's because the reliability of your conclusion depends on your accurate representation and reproduction of the underlying data; is that correct?

A I believe that is, in a very broad sense, correct. Obviously there is lots and lots of details that have to be addressed but, in general, that's true.

Q Now, you would never intentionally misstate or mistranscribe data from other source, would you?

A No.

Q That would be dishonest?

A Well, you might make a mistake. It's not necessarily dishonest. There's certainly the possibility of an error or misinterpretation and I wouldn't consider

that to be dishonest.

Q But you would not intentionally misrepresent or misstate data?

A No.

Q Any conclusions that would be drawn from intentionally manipulated data would also be dishonest; is that right?

A I don't understand the question.

Q If you intentionally misstated or someone else intentionally misstated underlying data and then were to draw conclusions from that intentionally misstated data, the conclusions would be dishonest; correct?

MR. LUNDY: I object to the form of the question. I don't know how he can testify about anybody else's state of mind and forming any conclusions.

BY MR. HOPP:

Q Can you answer the question?

A I'm having trouble with your concept of dishonest. I suppose that people have misrepresented data and done it intentionally, but that is not very common.

Again, I think it's possible people make a mistake or look at the data incorrectly, that they have a bias that influences how they interpret the data. I'm not sure I'd call that necessarily dishonest. In some

cases some of the data that has been collected has been collected in such a way as to create a certain impression.

They design the study from the beginning to get the results they want, and you can say that is manipulation of the data and could be labeled as dishonest. I have seen that done where the study design is so obviously flawed that they're not necessarily going to get data that can be relied upon, but you have to go in an individual case to, you know, reach the conclusion that dishonesty was the motivating factor.

Q If you were aware that someone took a published paper that had a value in it and then looked at the value and either intentionally transposed or misstated a follow-up paper that relied on the published data, that is grounds for some type of discipline or some type of report to a governing body?

MR. LUNDY: I will object to the form.

THE WITNESS: Again, I'm not in the business of judging the motivations of people and I have never seen anybody else do that either. There has been some talk in recent years about unethical research but we have to talk about it in an individual case to be more specific. I mean -- I'm just not sure how to answer your question.

BY MR. HOPP:

Q Have you ever encountered, either in published accounts or personally someone who intentionally misstated data?
A No, but what I've seen people do, like Dr. Wong did in his paper that he prepared in this case, where his study design is so flawed that he intentionally I believe designed the study to get negative results and had not analyzed his data in a way that I would say is consistent with generally accepted principles, and he built a bias into his research which, in spite of that, he still had some positive data, which he ignored, which I thought was ridiculous but -- I think due to his bias, I didn't consider it to be too honest.
Q We'll talk further about Dr. Wong as we get into it.
You rely on data that's not published, as well, at least in this report not published in peer-reviewed literature?
A There are reports that we have obtained that are studies that have been done and not published in the peer-reviewed literature.
Q And sometimes you rely on lab reports that have been generated, like the ERGO lab reports?
A Yes, we rely on studies we did on the individual plaintiffs in this case who represented the

exposed population.
Q Has any of the literature you rely on in your list of hundreds of reference, is any of that a lab report or unpublished lab report?
A The references are references and lab reports are lab reports.
Q Other than the ERGO lab reports, have you relied on other lab reports?
A We talked about a lot of lab results that are important and most important, other than the ERGO results, are the results of the environmental testing that's been done through 3TM, where they measure dioxins and PAHs and house dust and soil and the environment of the homes.
There's also the PAH adducts done by Dr. Phillips in the cancer research laboratory in England and the lab results done by Pacific Toxicology and lab results that we performed on the various plaintiffs we've seen.
Q And it's important that the lab be reputable, to use a good lab?
A Yes, it's important that you use a lab that gets accurate results.
Q And it's important that the lab is careful in how it handles samples?

A Yes.
Q And it's important that they have quality control and quality assurance procedures in place?
A Yes.
Q And it's important that the lab be able to show you it's QA/QC procedures?
A Yes, that's important.
Q And you rely on the lab to follow its QA/QC procedures; correct?
A Yes.
Q And a lab will usually have QA/QC documentation that it can provide with its report; right?
A Usually, yes.
Q In any event, you use a lab in part based on its representation for quality; is that right?
A Yes.
Q And a reputable lab provides a lab report for you; is that correct?
A Yes, provides a lab report.
Q The lab report should identify the task that the lab was asked to perform; is that right?
A Yes.
Q And the lab report should identify the sample collection procedures; is that correct?
A It should include the sample collection

procedures? That doesn't usually occur. The report doesn't include the sample collection protocols, no.
MR. LUNDY: I'll object insofar as all his questions assume that a request is made for that data and I think that -- he's implying that a request was made for all of that data, and with that false implication I'm objecting to the form of the question and whether a lab or not does that and -- I think they all do it, but I think your question is suggesting that we or Dr. Dahlgren requested all that.
Listen to his question closely and don't assume anything.
BY MR. HOPP:
Q A lab report from a reputable lab also sets forth the samples received and storage procedures; is that correct?
A A routine lab report doesn't include the QA/QC procedures, and those are usually done only on special request for most lab work. You don't get all of the QA/QC procedures, sample-handling procedures, chain of custody data -- all that stuff is available, if needed, but it's not part of the lab report.
Q A reputable lab will also provide a report showing its sample preparation procedures; correct?
A They will tell you if you ask what the sample

prep requirements and protocol are. These are standard operating procedures in labs. It's not included in every report because it's burdensome and a waste of everybody's storage space, if nothing else.

Q A lab report from a reputable lab tells you what analytical method was used; correct?

A Not always.

Q A lab report from a reputable lab will tell you what the results of its analysis were; correct?

A Usually they'll tell you what analogues they looked at and what the results are and what the normal ranges are and that's it. They don't give you all the rest of the stuff.

Q A lab report from a reputable lab shows you what the QA/QC procedures were that were used; is that correct?

A You asked that same question already, and I said that no, routine lab reports do not include the QA/QC procedures or the results of the QA/QC work done in that run.

Q The work you've done in this case or in other cases, is it important that the data you rely on be reproducible?

A By definition, if the lab data and the lab measurement you're taking is not reproducible, you would

not even do it. One of the things a lab has to do when setting up a procedure is to make sure it's reproducible and that the results on the same specimen will be in agreement within -- depends on the type of test, but within usually 5 to 10 percent, and sometimes lab tests are so variable that we even accept up to 20 percent variability, and you have to have reproducible results to offer the test in the marketplace.

Q Do you hold yourself out as a scientist?

A I think what I do is scientifically sound, yes.

Q Have you reached scientific conclusions in this case?

A I'm not sure what you mean by scientific conclusions. I've reached medical conclusions about medical facts in this case based on scientific evidence. Scientific conclusions? I'm not certain what you mean by that particular phrase.

Q Are the conclusions you've reached, the medical conclusions you've reached on the subject of causation for the plaintiffs in this case, is that the subject of your opinions, causation?

A That is the major issue we're addressing. I'm not disputing the diagnosis in most cases and you have fairly clear-cut medical problems.

The question I was asked to address is what is

the contribution of the chemicals arising from the wood treatment plant on these health problems, and that's the main issue, I believe medically, and not what their diagnosis is, and there may be some dispute in some cases about the diagnosis but I don't think it's primarily that. It's primarily an issue of causation.

Q Do you have a methodology that you apply in order to answer the question you've been asked to answer in this case?

A Yes.

Q Can you describe for us in general terms your methodology for answering these medical causation questions?

A Sure. The first issue one has to establish is what I'd call generic causation, and that's are the chemicals in question capable of causing a health effect and, if so, what type of health effects are they known to cause based on studies done in animals or humans, case report studies or epidemiological studies, mechanism studies, in vitro studies, and there's a whole host of different ways one can identify the toxicity spectrum of the chemicals present in this case or any case.

The second major issue is to evaluate the individual patients or in this case plaintiff or subject of the lawsuit to determine what their health problems

have been, what their medical history, occupational history, residence history, family history, lifestyle, habits, hobbies, as much information as one can obtain about everything that has to do with their medical history and lives.

At the same time you collect that information, you need to collect information of exposure, and what does that person have in the way of a historical exposure history, and they describe where they lived -- in this case we're talking about residential or environmental exposure, and how long they lived there, and how close to the source, and whether or not they had opportunity for additional exposure such as, in this case, people with burn-treated wood inside their homes, play in the ditches during the summer time and be exposed to water or sediment.

Even in this case some of the children would go and climb on the wood in the wood treatment areas, as the wood was curing or being allowed to be stored for a period of time before it was shipped. Collect as much information about that individual's health and exposure data by history.

And then, of course, look into whatever data there was on exposure from the facility, as it relates to the subject. What was known about the discharge of the

chemicals into the air, soil, water and other products coming off the property that the subjects we're talking about come in contact with, so we get as much documentation of the chemicals and chemical levels and that would occur.

Once we have information about dosage, we want to also look at, as I said and repeat it to make it clear, the differential diagnosis. If the patient has a lung cancer and the issue is was it due to the exposure of the dioxins and PAHs from Koppers versus their own exposure to say cigarettes smoke they may have been exposed to either from personal habit of smoking or secondhand smoke from other family members.

In that case, we might collect data not only from the patient but from other family members who would have known about the person's exposure. In the case of children who can't speak, we have to collect information about second-hand smoke from, let's say, family members or friends of the family that knew about it.

Putting together all the causative factors, we apportion which factors are the most persuasive and important and potent to address the issue of causation.

We also need to review the medical records to be sure the history we obtained is accurate and backed up by what the doctors have actually found, insofar as it is

possible. Frequently medical records, particularly for things that happened in the past, are somewhat difficult to get because they're destroyed, but we try to get as much information as possible.

Then we can address the issue of specific causation when we've established that the chemical can cause certain diseases, does the person's specific illness match with what's know about its generic causation and if they had a sufficient dose of exposure to cause that illness or contribute significantly to that illness or health problem. So that's in a general way how one approaches this question.

Q I want to ask specific questions about your method.

Part of what you described is collecting data on exposure and the plaintiff and collecting all of the available data; correct?

A Correct.

Q And you reviewed all the relevant published literature?

A Yes. I indicated under generic causation that one would look at the medical literature in detail as to what those chemicals are capable of causing.

Q Is it important to you that your conclusions can be tested and someone else following your method can

reach the same result?

A Yes, they would be transparent and very clear on what the basis of the opinion would be.

Q Is it important to you that the method by which you collect and analyze your data has been subject to some type of peer review?

A Well, I guess I would have to say that the methodology that I have described is methodology used by others, whether that has been peer-reviewed -- I mean, I think somebody did write an article about peer review and causation -- I can't remember the name of the author, but there have been a few articles written and published in various journals about an individual's opinion on how they approached the causation question.

I don't remember the authors' names but, in general, what I described to you is similar to what others use for finding causation and has been a subject of peer review.

Q You've published at least one health assessment of population of people living near a wood treatment plant; is that right?

A Yes.

Q And that health assessment was subject to peer review?

A Yes.

Q At least twice?

A Yes.

Q Did you follow the same method in this case, the same as in your published article?

A I think so, although we have more higher levels in this case than we had in the Columbus, Mississippi case. The levels are off the charts in Grenada, in the house dust and soil and the people there, significantly elevated values and really horrendously high values, particularly the house dust values, which are so high as to be, I think, capable of causing harm and really incompatible -- people I don't think should stay in those homes and should be remediated or torn down and need to be moved immediately because they're experiencing irreparable harm by staying where they are.

Q Now, we're talking about method and you said there are distinctions between Columbus, Mississippi and Grenada, Mississippi but is your answer that you followed the same method in both cases, that is collecting and analyzing?

A We collected and analyzed and had more data in Grenada. We had the DNA adducts that we didn't have in Columbus, and these document the presence of high levels of PAH adducts which we did not have in Columbus.

These PAH adducts predict an increased risk of

cancer in these folks and, therefore, the exposure is ongoing. The PAH adducts are usually only reflective of the previous three to six months of exposure, so these people have, I'd say, qualitatively higher exposures and quantitatively higher exposures and at very, very high risk and those exposures are ongoing and, therefore, this is something of a medical emergency in my opinion.

Dr. Wolfson and Dr. Sawyer and Randy Horsak and Devrage Sharma, all the other experts I've spoken to about this are in agreement with me that this is a medical emergency and that these people are living in a highly dangerous environment and this needs to be addressed on an urgent basis.

Q Have you shared the concern with the Mississippi Department of Public Health?

A No. I've shared them with Mr. Lundy and his associates and I believe they have communicated those concerns to the defendants. Frankly, the idea of going to the Mississippi Department of Health -- was that the question or the environmental quality --

Q Any public agency that would be responsible for taking care of these people --

A Well, that would be wonderful if they had a track record of doing anything. I guess you can consider contacting them and that's an idea that didn't cross my

mind just based on the history with health departments in the last 30 years. They usually don't respond, even when they should, but we can send them a letter and ask them to look into this matter.

Q The same question for any county authorities, Health Department authorities?

A No.

Q Does your method, the method that you described, have a known or potential error rate?

A I'm not sure what you're talking about. An error rate to a method?

Q Yes.

A I don't know how you put a quantitative value on a qualitative process like that.

Error rate for an assessment protocol?

I guess what you're saying is always a hundred percent accurate versus 50 percent accurate?

Q Has it been tested, does it have an error rate?

A You'll have to clarify what aspect of the generic causation assessment. The rate of collecting the generic causation data? It's there or it isn't. It's one of those all or nothing.

Either the generic causation data is there or it isn't. Either the patient's history is there or it isn't. It's not like you can say that the likelihood

that the generic causation assessment is correct is 75 percent accurate. There is no way to say that. Either they have it or they don't.

Q So we're clear, I'm not talking about the generic causation assessment. I'm talking about your entire methodology which includes generic causation assessment, all the data you stated you collected and reaching a conclusion regarding a particular plaintiff with respect to either causation or increased risk of future disease.

Does that method that you painstakingly laid out for us, albeit in generic terms, have a proven error rate?

A I don't think that the term error rate would apply to what I described. You can apply the term an error rate to the measurement of the dioxin in the blood. You can apply the term error rate to certain other aspects of the thing. Various tests can have error rates but the overall methodology I don't think the term error rate applies.

Q Let's talk in more detail about the data you collected. Is some of the data you collected data on the source of a potential exposure?

A The fingerprint, the pattern of the dioxins, matches the dioxin exposure that you would expect from a

pentachlorophenol source. So that data collection did address the issue of identifying the source.

Similarly, the 3TM data that was collected by house dust also showed the same pattern, which is the predominance of the larger chlorinated species of CDD and the hexa CDD species. That's also fingerprinted to the pentachlorophenol.

Q The general idea being that you can't have exposure without a source, and you have to identify the source of the exposure that you evaluate; right?

A As best you can, yes. There is obviously times when you're not sure, but in this case it's clear that we traced the dioxins to the pentachlorophenol based on the fingerprinting that I mentioned.

Q You also need to collect data on exposure; correct?

A Yes.

Q Did you collect data on exposure in this case?

A Yes.

Q You can't have a toxic effect without the exposure to the toxin; correct?

A You have to be careful and there's a great deal of variability in people's response and some respond to very low levels.

In general what we say in toxicology is that

the dose is important and it has to be a sufficient dose to create the effect and taking into account even the most sensitive sub-populations, such as in-utero fetuses that are growing in the womb, which are probably the most susceptible to toxic effects and various agents, and the dose in those cases were generally found to be quite lower than say the effect it takes to have a toxic effect on a full grown healthy adult.

Q Is there a difference between exposure and dose?

A Well, you can have an exposure. If you have exposure there is some dose associated with that. It can be a high dose, medium or low dose, but if you had exposure, you've had a dose. It's a question of how long and how much but still if you had exposure, you had a dose.

Q And if you have not had exposure, you don't have a dose?

A Correct.

Q For example, grain alcohol is toxic; correct?

A It can be toxic. It depends on your definition of toxic and dose that it would take to create any given effect.

Q It can have an acute or chronic effect; correct?

A You can put a drop of grain alcohol on somebody's skin and it probably would not have any adverse effect. You can take a quart of grain alcohol and drink it and it can kill you. It all depends on what you're talking about.

Q We keep hinting around this. You've identified a sort of common theme with toxicology.

Are you familiar with a man by the name of Paracelsus?

A Yes.

Q And he had a common phrase?

A Yes.

Q What was that famous phrase?

A Dose makes the poison.

Q Let's go back to the grain alcohol example. What I'm looking for is something simple. If you don't open the bottle, you don't have exposure; correct?

A Okay. As long as you didn't break the bottle or it didn't seep out, there is no exposure.

Q If there is a barrier between you and the source of exposure, you won't have the exposure; correct?

A That's by definition.

Q That's what we're talking about, definition. And a dose is the amount of the toxin which crosses some membrane and gets into your body; correct?

A Yes.

Q And it can be a large or small dose depending on the method of how the does is administered and how much; correct?

A Over a period of time, yes.

Q In the example you've indicated, if someone opens the bottle of grain alcohol and drinks it, they can have an acute alcohol poisoning and die.

A If they drank enough and fast enough, yes.

Q If they drank enough over a long period of time, say years, they can have liver cancer; is that correct?

A That's a possibility, yes.

Q Liver cancer is a known toxic end point of grain alcohol; correct?

A Yes. Alcohol use is associated with increased risk of liver cancer.

Q Did you collect data on dose in this case?

A Tried to, yes, in every case.

Q How did you try to collect data on dose for this particular case?

A Most importantly was to obtain information about the patient's personal history -- where they lived, what they noticed, what they smelled, the symptoms they experienced in response to exposures that occurred from

the plant.

Whether they played in ditches or burned treated wood in their home or played in the ties and walked into the plant and got close to the treating cylinders or worked in the plant or other plants.

But focusing on the dose, the issue of the exposure to the Koppers PAHs, pentachlorophenol and dioxins, personal history was important and looking at other data such as the estimated materials coming out of the plant, what the contractions were in the house dust in homes and what the company's practices were and the tons of material they used, what kinds of pollution control systems they had to try to reduce air emissions or discharge of water and chemicals into the soil and ditches and into the home area where these people lived.

Also reviewing the data collected by others, such as 3TM and Sharma's data collection of what was done in the plant and likely to be the quantitative aspects of the exposure that occurs in the vicinity around the plant.

So dose is the result of all of these assessments. Doing a retrospective reconstruction is somewhat difficult, but it's clear when you describe living in a neighborhood and smelling the creosote and noticing the particulate material building up inside the

homes, playing in ditches and playing on the ties with skin absorption, inhalation through those routes, one can get a good qualitative sense of high, medium, low exposure and was their exposure different and unusual compared to a general population in a certain class of compounds.

In this case dose is, I think, fairly easy to say that it was high, and the study we did in Columbus, Mississippi and the study done by McGee in Kansas City, Missouri shows that even people who don't experience as high a level as these people in Grenada did have health effects attributable to treatment plant environments.

We're talking here in Grenada about an exposure that I consider to be helatious and it's incredible that these people live right up next to the plant that processes millions of pounds of toxic chemicals that we talked about, discharging a huge amount into the air and soil and water around the plant, contaminating this area, like I said earlier, to such a degree that it's truly a health hazard to live there right now.

Q During the course of that extensive answer, you hit upon a distinction I want to explore.

You used the term qualitative. Remember that?

A Yes.

Q Is there a difference between qualitative dose

information and quantitative dose information?

A Well, there is such a thing in toxicology of what we call high, low and medium exposures. Most of the literature we have on occupational and environmental exposures in the last hundred years of research in this field has used qualitative dose.

In other words, we'll take a job description that we'll say is a high exposure like, for example, in coke ovens. Workers that are on top of the coke oven are considered highly exposed. The workers on the ground or in the vicinity but not on top of the cokers are considered to have moderate exposure, and then workers in another category, in another part of the steel plant where making the coke is going on, is considered low exposure.

Then you look at the health risks in the three groups, and that's the way most research has been done. It's qualitative. We can't go back and reconstruct 50, 30 or 20 years of exposure. We can't do that and we have to make estimates. And that's what we do in all these cases is make estimates.

We get as much quantification as possible, and we've done that in this case, but we have to also start talking about that this person was born and raised in this environment and lived all their lives there and got

therefore a very high dose -- we can't say how many micograms while in the mother's womb, but we can say they were having exposure and we'd classify that exposure as high.

Q *These qualitative exposure categories you* described are a way of describing dose in the absence of physical measurements of the environment that someone was in or physical blood, urine samples?

A In the absence of Koppers not having a fence line monitor when measuring the chemicals on an ongoing basis.

Q These high, medium, low exposure categories are a surrogate for dose information; correct?

A They're a surrogate for dose and give some way to classify the dose that someone has experienced based on all these factors I mentioned.

Q You talked about, in your extensive answer a moment ago, a quantitative method for obtaining this information, in particular you mentioned 3TM; is that right?

A Yes.

Q Is that Randy Horsak's company?

A Yes.

Q And Devrage Sharma?

A Yes.

Q And he also collected quantitative information; correct?

A *He did quantitative measurements or estimates* based on how many pounds of the chemicals --

Q He's the modeler?

A Yes.

Q Horsak went out into the neighborhood and collected dirt and dust and things like that?

A Yes.

Q And took it to a lab and you have a lab report or a series of lab reports from Mr. Horsak that describe data collection, analytic procedures, etc., that give you the results that Mr. Horsak obtained; correct?

A Yes.

Q Can you use that quantitative information in your assessment of causation?

A Yes, that's part of the assessment.

Q How do you use the numbers, physical numbers that someone collects and use them to evaluate causation?

A Well, as I think I indicated, they're an indication of current exposure, the levels in their homes of PAHs and dioxins that can be used to estimate what their current dose is, what they're being exposed to as we speak, living in those homes.

In most all the homes, they're above safe

levels and in most of the house dust samples and soil dust samples obtained in that neighborhood in the homes of these plaintiffs we're talking about here today, exceed the Mississippi Department of Environmental Quality's cleanup standards for dioxin.

In some cases they reach levels that would be likely to cause acute illness and not just chronic illness, the levels are so high. But the ingestion of the dust on their fingers and in the food and inadvertent ingestion and absorption and inhalation levels of these chemicals would be above safe levels, above the minimum risk levels that have been established by the ATSDR for these classes of compounds.

MR. HOPP: Can we take a quick break and bring Mr. Collins into the room.

(Recess.)

MR. HOPP: We're back on the record.

Q Dr. Dahlgren, when we took a break we were talking about the difference between qualitative dose and quantitative dose measurements in this case, and I believe your response covered minimum risk levels and the extent to which various regulatory thresholds are exceeded in the neighborhood surrounding the Grenada plant?

A Yes.

Q Other than looking at the data that Mr. Horsak collected and comparing it to regulatory levels, have you done anything else to identify quantitatively what the doses are for the seven plaintiffs in this case?

A We have measurements of PAH adducts and dioxins in similarly situated people and exposed on the same lines as these folks in the Beck case and found them to be elevated.

As I said earlier in the discussion, the patterns were compatible with the materials coming out of the plant, so even though we've not done it on the individual plaintiffs, we can safely assume that if we were to make the blood measurements, they would be similarly elevated to those we have measured.

Q So we're clear, you have had some people from Grenada, you have had their blood tested for dioxin and PAH, DNA adducts; correct?

A 29 people, yes.

Q And that doesn't include the 12 plaintiffs in the Beck case; correct?

A Correct.

Q Why is that? Why did you not test the 12 in the Beck case?

A I think it's an economic issue. With the dropping dollar, we're about $2,000 just to do the

dioxins and furans, and it's just because of the expense. I forget the price of the DNA adducts, but this is not yet been done, and I would like to do it but it's just expensive.

Q And the dropping dollar is at issue because both labs are in Europe?

A Yes.

Q The laboratory that did the dioxin work is in Germany?

A That's correct. And even if we used the AXYS lab in Canada, which is a reliable lab, the expense is still very high without the dropping dollar. The Canadian dollar is stronger than the U.S. dollar, relatively speaking, but it's still approaching $2,000 to do the studies that we need to do up there.

MR. BAILEY: $2,000 U.S. money?

THE WITNESS: Yes.

BY MR. HOPP:

Q And the DNA adduct lab you used is in England?

A Yes.

Q Let's go back to the notion of quantitative dose measurements.

Are there equations or scientific formulas that exist that you can use to take the data that Mr. Horsak collected and calculate the risk for any of the 12 in

this case?

A Yes, I think you can construct a dose metric that would allow you to estimate the cancer risk associated with the current levels of exposure.

Q And that's something done fairly frequently, that is quantitative risk assessments for exposed populations; right?

A That's correct.

Q Have you done that in this case?

A No, Dr. Sawyer has done that in this case.

Q Do you rely on Dr. Sawyer's quantitative risk assessment?

A Yes, I believe his risk assessment is done properly.

Q Now, I've been through your report and I don't see any mention of Dr. Sawyer's risk assessment.

Do you cite it in your report?

A No, I didn't see it. At the time I put my report together, he was putting his report together and I didn't have the actual data.

He told me in general that everybody in the whole 12 had estimated risks that were unacceptably high but I didn't have his actual numbers for each person until recently. Now I have that data on the 12 people.

Q Now that you have it, do you believe it

supports the opinions you gave without having it?
A Yes. It's entirely consistent with what I had estimated before, which was that these people have high exposures, based on the blood levels we did and the environmental testing and the subjective histories the patients gave and based on what we know on the amount of materials used and what we know about the epidemiological studies. All of it fits together and it's not at all surprising that Dr. Sawyer's data is consistent with all of that.
Q Now, we talked at length about how you collect data and the different types of data you collect.
Is the idea that you try to tie the exposures and doses you measured back into the specific conditions you see in the plaintiffs?
A Yes.
Q To do that you need a diagnosis on the plaintiffs?
A Yes.
Q Did you do a diagnosis for each of the plaintiffs in this case?
A Yes. As I stated, I relied on their own doctors for diagnoses of cancer and other serious diseases and I didn't make an independent diagnosis.
Now, in some cases we noticed the presence of

various respiratory complaints and various neurological and skin and other types of symptom complexes, if you will, that have not resulted in a diagnosis by their treating doctors.
In some cases they diagnosed asthma or bronchitis or pneumonia and in other cases the patients had the symptoms compatible with those diagnoses, and we can safely say that there are some patients where the diagnosis would be made based on their symptoms that they described to me or the findings on the tests we did or done by others. So there is a variety of different diagnostic groups here.
Q And one of those is the preexisting diagnoses you find in the medical records?
A Yes.
Q And you actually visited with each of the living plaintiffs; is that right?
A Right.
Q And did you reach additional independent diagnoses not reflected in the medical records?
A As I said, there is certain symptom complexes that allow themselves to be made into a diagnosis, and I have not necessarily rendered a diagnosis from those symptoms and in these cases, but it's possible to do so.
Q Why have you not rendered diagnoses based on

the symptom complexes you observed in these plaintiffs?
A As I said, I could, but in my report I laid out the symptom complexes that I believe have been aggravated by the exposures and not put a label on those symptom complexes.
For example, many of them had complaints of chronic productive cough or wheezing. We can diagnose bronchitis from the chronic productive cough and asthma from the wheezing but I have not done that. There is no particular reason for that, other than it doesn't make that much difference if we're describing the illness and health effects. You don't have to necessarily put a label on it. We're describing what the problem is.
Q Is there some reason why you didn't put a label on it? Is there something stopping you from saying Mr. Jones has --
A No. There's nothing stopping me from saying Mr. Jones has bronchitis and in some cases we've said they have bronchitis based on their symptom complex but not in every case.
Q I'm sorry to make you go over this again. There's nothing stopping you and you have not done it.
Is there a particular reason why you have not diagnosed these plaintiffs based on their symptom complexes?

A We can go through individual patients and talk about labels for their symptom complexes, if you wish, and I just feel for purposes of describing the patient's injuries, it was better to describe, especially in those cases where the diagnosis had not been made in the medical records, simply describe what we've been told is the clearest and most simple way of communicating that information.
Q Now, once you collected the data and the environmental data, the qualitative exposure data you described earlier and the diagnosis or the symptom complexes you described, at that point you have to frame the question for each plaintiff; is that correct?
A Yes. You have to frame the question for each plaintiff.
Q And you did that for each one of the plaintiffs?
A Yes.
Q And what's the next step? You have the data and framed the question, what's the next step in your method?
A Well, the way you're constructing it, the next step is to look for other factors that could contribute to their health problems, and these are occupational, environmental, lifestyle, family history, hobbies, any

and all factors that would be important to cause those symptoms in that individual patient -- so-called differential diagnoses, although in a sense what we're doing is differential causation assessment rather than differential diagnosis, since we're dealing with a given health problem.

And what we want to know is what is the causative effect. They have an irritated bronchial tube and cough, wheeze, short of breath, have chest pain, and what's the reason they have those symptoms? If they happen to be a heavy cigarette smoker or a life-long history of asthma before moving into Carver Circle, I'd talk about someone who had aggravation of a preexisting problem rather than something where the exposures were the primary cause.

Q And we'll go through the individual reports on the individual plaintiffs in this deposition, but have you documented that effort for each one of the plaintiffs in this case?

A Yes. We have an extensive questionnaire and history and evaluation of their depositions and their medical records have been made available and all these things are looked at with the concept in mind of trying to find out any and all of those factors that are important.

Q In your experience, generally, have you ever had a plaintiff in a lawsuit that presented to you and have you ever reached the opinion that the exposure, the issue in the lawsuit, was not the cause of the plaintiff's problem?

A Sure.

Q How many times have you seen that?

A Many hundreds of times.

Q Did it happen in this case?

A Some of the health problems are not related to exposure, yes.

Q And are those laid out in your summary, the health problems that the plaintiffs have that are not related in exposure?

A In my conclusions I did not discuss this and I talked about things that were related and not what's unrelated.

Q So anything not in your conclusion is unrelated?

A Well, I think so. I might have made a mistake in some cases, but I tried to talk only about the issue stuff, what I thought would be caused or aggravated by the exposure.

Q Now, once you've collected the data and collected the diagnosis or the diagnostic indicators and

done your differential causation assessment, what's the next step in your methodology for assessing causation?

A We've already assessed their exposure to a chemical that's known to cause the health effect at a sufficient dose and taken into account all other possible causes and now reached the point to reach a conclusion about the patient's health problems and what role the exposure had in causing or aggravating those things and that's my conclusion phase.

Q I want to talk about your use of epidemiological literature to assess causation. We talked earlier that you used epidemiological studies in your general and specific oausation assessments; do you remember that?

A Yes, they are used.

Q Can you tell me what an epidemiological study in this context is designed to do?

A To see whether the exposed population has a different pattern of illness than a comparison population or control group.

Q Now, you can't use an epidemiological study to prove that a particular person got a particular disease from a particular exposure, can you?

A You have to look at groups of people. However, you can use that for generic causation purposes and, in

other words, you can say that since this population has a higher prevalence of cancer and you know from other information, for example, animal studies, case reports, other epidemiological studies of similarly exposed people, all showing that cancer is one of the effects that this chemical has.

And then Joe Blow lives in the environment, significantly close to it, I think you can make a conclusion in the individual case that there has been a contribution to an increased risk of cancer from that exposure. So epidemiologic data is helpful in establishing cause and effect relationship in that context.

Q But the point you made earlier is that epidemiological studies look at groups of people and compare exposed to unexposed populations?

A The comparison groups have all the other risk factors. They smoke the same, the same family history and exposures to diet and same exposures in their work activities. The only difference between the two groups is this exposure, and that's the purpose of the study -- of course it's the ideal and it's hard to make it perfect but by using large enough numbers, you get valid results from such techniques.

Q Let's for a moment focus on the difference

between general causation and specific causation and you've used the terms several times.

Can you define the difference between general and specific causation?

A General causation is looking at all the data about the published and unpublished effects of the chemical in either an animal test system, in vitro or in human models, and that includes all the data, case reports and epidemiological studies and all the information that one brings to bear, even similar chemicals, not identical but similar in their action. That's generic causation. That the chemical X is capable of causing disease Y, and that's the basis for that.

Q So what's specific causation, as distinguished from general causation?

A Where an individual patient, subject or plaintiff has been exposed to environment X and has disease Y.

Q When you're doing your general causation work and looking at epidemiological studies to evaluate general causation, it's important that the studies you look at be relevant to the exposure at issue?

A Yes.

Q If you look at a case involving solvent exposure, you will not find proof of general causation in

at studies involving that class of chemicals and there is a lot of other settings in which PAHs occur and give us relevant information about the health effects of that compound.

Q So if a compound is made up of constituents, you can look at studies that focus on constituents; correct?

A Yes.

Q But you don't want to ignore studies that focus on that compound; is that right?

A No, you wouldn't ignore them.

Q And why not ignore studies that focused on the compound at issue?

A Because it's relevant.

Q And when you're looking at epidemiological studies for proof of general causation, you want to look for studies that examine the same disease or diseases you're interested in; right?

A Well, you want to look at studies that looked at all of the diseases. Certainly. You don't want to preclude -- obviously, you want to look at all the diseases that might conceivably occur.

Q Let's take another example. If you have a discreet case involving TCE and kidney cancer, you want to look at studies that address TCE and kidney cancer;

studies of metal exposure; is that accurate?

A Correct. There is no reason to think that those two classes of chemicals act similarly and would act differently, although both can affect the respiratory tract, but you would not use the study of chromium exposure to look for generic causation in a group of patients exposed to organic solvents.

Q You've done chromium cases; correct?

A Yes.

Q And you've done solvent exposure cases?

A Yes.

Q And in each instance you look for the studies the closest as possible to the exposure in your study; correct?

A Yes.

Q And if the study looks at the exact same chemical or compound you're looking at and a similar or identical exposure condition, you're actually going to want to use that particular study; is that right?

A Yes. You want to use all the studies that are relevant. It doesn't have to be identical exposure. What we want is for the chemicals at issue to be similar or as close to similar as possible.

With creosote you have a mixture that contains PAHs, the most toxic element in creosote, so you can look

right?

A Yes, you want to do that.

Q And if a study looked particularly at TCE and skin cancer or brain cancer or some other organ system, that's not as relevant; correct?

A Well, it's not relevant to the question of whether or not kidney cancer is shown to be associated with TCE. It's relevant whether TCE is known to have a carcinogenic effect on whatever tissue it is.

This whole concept that you can identify all of the cancers associated with a given chemical by the most common ones that have been described up to now in the literature would be a mistake. You need to look at all of the cancer.

I notice a number of studies have done that, not looked at all the cancers and picked, let's say, the cancers previously reported to be present in higher concentrations or higher prevalence and that would be a mistake, I think, because there is a great deal of variability in how populations respond and individual variations and different exposures and different time frames.

So you want to keep an open mind about that and certainly there is no reason to think that because a chemical was predominantly causing kidney cancer in a

population, it's not capable of causing a disease in other organs.

Q Are you familiar with the concept of target organs for chemical carcinogens?

A Yes.

Q Describe for me the concept of target organs.

A Well, there are chemicals that cause a cancer in a specific organ at higher concentrations and let's take asbestos which we talked about earlier.

The most common cause of death in an asbestos worker is lung cancer. Why is that? Because asbestos fibers are present in the lung in high concentrations and most of the fibers of the body are in the lung and it makes sense that you will have most of the cancers in that organ.

So a lot of the research that was done on asbestos was focusing on lung cancer and that doesn't mean it didn't cause cancer of the organs but meant that it caused mostly cancers in that organ.

Q Are there organs which particular chemical carcinogens do not target?

A Yes. There is fewer cancers, for example, in the bone and bone tends to have fewer cancers overall than say most other tissues in the body, and the reason for that is probably relatively low concentration.

Various carcinogens, for example, asbestos doesn't get in the bone, and there is relatively low concentrations of most of the carcinogens in the bone.

However, there has been reports of increase in connective tissue with dioxins, for example, and the reason for that is unknown but bone is an example of something with relatively low rates of cancer.

Q But when you're doing a study like the one you've done in this case, you want to identify the relevant target organs and obtain all the studies that look at the exposure at issue and the target organs at issue?

A Yes. And what I did in my report is try to give all the relevant studies about each of the cancers and health problems that have been published on that subject.

Q Do epidemiologists use what is called ICD codes for diseases?

A ICD stands for international classification of disease, and it is used. ICD9 codes is one way of doing epidemiological studies because you can quickly go through a large database and identify the various types of cancer.

Q And when you evaluate an epidemiological study for the purpose of doing a general causation analysis, do

you look for particular ICD codes to see where the cancer is elevated?

A I don't usually look at the codes and look for the description of the cancer. ICD9 codes are a way it can be done but I don't usually memorize or use the ICD9 codes.

I look at lung cancer and there are several types of ICD9 codes for lung cancer and different cells types, and that doesn't help us in, for example, asbestos. It causes several different kinds of lung cancer and there is no benefit in breaking it down to the different subtitles.

Q Let's talk about hematolytic cancer. Are there different ICD codes under the general classification of hematoseptic cancers?

A Yes, quite a few.

Q Are those relevant? Don't particular toxicants cause particular types of hematolytic cancer?

A Benzene has a very strong association with certain types of leukemias, acute leukemia being the most prevalent type. Benzene is associated with several other of the hematologic and lymphatic leukemias. It certainly is something you can do but it is -- it is not necessarily relevant to our discussion, for example, in this case.

Q Let's move on. You mentioned different types of studies and animal studies and case control studies and case reports, etc.

Is it true there are different types of epidemiological studies?

A Yes.

Q Are some considered more powerful in detecting associations than other?

A Yes.

Q Which is better?

A Where you have good exposed data and classify you're exposed group accurately. Unfortunately, it's rare that you have good exposure data, historically anyway. So the biggest problem with most studies is they don't have good data on exposure parameters. That's a general point.

Q Let's take a specific example. A clinical trial is a type of epidemiological study, isn't it?

A Yes.

Q And you're studying a particular dose of a particular exposure on a particular body system?

A Yes. When you're doing a pharmacological study, you're comparing a group of patients with the same diagnosis. Part of the group gets the medicine and the other part doesn't. You may have different doses

depending on what you're doing.
Q The advantage there, as you indicated, is you know precisely the exposure level; correct?
A Yes.
Q And you can control for that exposure level, that is you can control your population to determine whether they have other risk factors for the item you're studying; correct?
A You design your study so that you have an equal number in the two groups of all the risk factors and you don't -- you want to isolate the one difference between the exposed and unexposed group, what you're administering.
Q And clinical trials are particularly good at that; right?
A It controls variables pretty precisely because of that.
Q Is the size of a study population an indication of the reliability or quality of a study? Is a larger study better than a smaller one?
A No; especially if you dilute the effect like Dr. Wong did in his study where you took every employee who ever worked in any of the six Koppers plants and threw them into the study.
Someone could have worked there one day and

worked in the office for an hour and been included in his group. You get large numbers, but you're getting a lot of what we call misclassification bias and, you know, you can have huge numbers, and a lot of those studies done by Dr. Wong and others for industry do that. They have 5,000 people in their cohort but the exposure of most of them was not relevant, and so they're including people in there that dilute the effect of what you're looking for.
Q So you're saying as a general matter a larger study is not, generally speaking, better than a smaller study?
A I didn't say that. You said were larger numbers automatically better and the answer is no.
Obviously, when you have larger numbers and good exposure data, you can detect smaller differences. For example, Dr. Selikoff who studied the asbestos workers was able to show that not only were lung cancers and mesothelioma in excess, but kidney and pancreatic and colon cancer and a whole variety of other cancers were also increased in proportion of the increased dose of asbestos, making the point that it's a multi-organ carcinogenic agent, and by having a very large population that he studied prospectively over time with good exposure data, he was able to demonstrate some of the things that were not possible to demonstrate with smaller

numbers of people.
Q Assuming that the quality of the exposure data is equal, wouldn't a larger study have more statistical power than a smaller study?
A Yes.
Q And the type of exposure data you collect, that's important with respect to the quality of the study, one with better exposure data is a higher quality study than with worse exposure?
A Yes. That's the biggest problem with environmental and occupational exposures, overcoming misclassification problems.
Q And one of the ways you can collect exposure data is to go to the facility and measure it and measure what people who worked there were exposed to?
A Well, with creosote it's well shown now that you have to do worker biological monitoring in order to assess exposure because air monitoring is useless.
So all these studies done on air monitoring are a huge waste of time, and most of the exposure workers get in the creosote industry is from skin absorption.
For example, in the case of Dr. Wong's study where he's looking at individuals in the creosote industry and estimating exposure based on air measurements, which I think was done traditionally, you

can't rely on that.
Dr. Borak did a study for McGee, published a couple of years ago, that showed that air levels are very low but the level of one hydroxipyrene in their urine is very high and that's from skin absorption and the creosote council just released their results where they did detailed studies of workers in the creosote industry and found the root of exposure was skin absorption and not through the air.
In fact, Dr. Heikkila in Finland did the study and found a similar result where it was through skin absorption. There were several other studies done, and I don't recall the authors, that made the same point, so you need to have accurate exposure data, and in the case of the creosote industry, that's important, the biological monitoring.
Q We were talking generally about exposure data and all I really want is a general idea. If you have better more reliable exposure data, you're likely to have a better more reliable epidemiological study?
A That's true.
Q If you can actually go to the plant or take a blood sample or some biologic monitoring of the people you're interested in, that's more reliable than mailing a questionnaire?

MR. LUNDY: I object to the form of the question. Your question assumes that the plant is operational, that there is treated ties in place and assumes all these criteria that have to be in existence which there is no proof that any of them are in existence in studies the industry has done. So you're making a lot of assumptions in your question, and I don't think this witness can answer.

MR. HOPP: To be fair to me, you're talking about creosote and I'm talking about epidemiologic studies.

MR. LUNDY: So you're not talking about creosote?

MR. HOPP: We're talking about epidemiology in general, including creosote.

THE WITNESS: Well, the better your exposure data, the better your study is going to be. I would agree with that.

BY MR. HOPP:

Q You're familiar with studies that studied exposures by way of mailing questionnaires? You read some of those, haven't you?

A Mailing questionnaires have been used to assess populations and it's the most cost-effective way to collect information. I think they have validity and it

depends what questions they're asking and how they're -- what the purpose of the questionnaire survey is.

I found personally it's better to administer a questionnaire than to send it to someone and have it filled out on their own and, having said that, there is still some value in a carefully done questionnaire study.

Q And I'm certainly not arguing and would never argue the point that there is no value. But we're talking about ranking of epidemiological studies in order of quality, and you've identified certain aspects that make an epidemiological study have more quality than another one, and one of those things you've identified is exposure data, and there are different ways you can collect exposure data; right?

A Yes.

Q And how you collect exposure data affects the quality of your study, doesn't it?

A Yes. The quality of the assessment does effect the quality of your study.

Q Now, there are other studies you might look at, including animal studies; right?

A Yes.

Q And animals are different from humans and there are biological differences between the species; correct?

A Yes.

Q And animal data may have some relevance but what happens in a mouse given a particular dose of TCE is not the same as what will happen to a person given the same dose of TCE. Is that a generally accurate statement?

A I think it's generally accurate that there are differences between mice and men.

Q I read a book by that title. And there is also case reports, individual case reports that one might look at?

A Yes.

Q That's a type of study you'd throw into the mix, isn't it?

A Absolutely.

Q Again, that's on the quality ranking an individual case report is lower than say a closely controlled clinical trial; is that right?

A It depends on what end point you're looking at and what you known about the exposure.

For example, when angiosarcoma was discovered by Dr. Kreech (phonetic) in Louisville, Kentucky, looking at a cancer that occurs like one out of a hundred thousand patients with cancer and he had three in one practice, and they all worked in the same plant and did the same job, he didn't need a carefully controlled study

to know there was something wrong with that and he published his study as a case report but it led to the reduction of exposure to vinyl chloride from a thousand parts per million to one part per million.

So no epidemiological study was needed to make that conclusion because the data was so overwhelming. The likelihood of these three people getting cancer from some other causes is infinitesimal. So you didn't need a big fancy study. If you have three patients with lung cancer, it may be a bigger problem. It's a common disease or three patients with diabetes, it's not so clear.

Q First of all, angiosarcoma is a unique circumstance, that type of thing doesn't happen frequently, what happened in the case of angiosarcoma?

A Well, there is other examples where you have a rare disease. The mesothelioma coming up in the original reports by Wagner and showed this rare disease that was only occurring among these asbestos miners and made the point. He didn't have to do a big control study and he knows in the general population this disease is rare and finding a bunch of them, the numbers were so outlandishly high that that was enough to establish causation.

Q But for the more common cancer, like colon rectal cancer, an individual case report is not going to

be as statistically powerful as a closely controlled epidemiological study --

A In that case, I stated you need a comparison study to find out if there is a higher prevalence in that population.

Q Is there a method, is there a quality instrument that you apply when you look at the epidemiological studies to rank them or order them in some way in terms of quality?

A It depends on what the questions you're asking are but, in general, exposure assessment is important, selection of the population to avoid misclassification, collection of your population so that you have all the various variables, for example, cigarette smoking or dietary factors or other environmental exposures are well characterized so when you match your population, you match it appropriately.

Q The question I'm getting at is, is there a method you can describe for me that you use when you gather -- let me back up.

You do a literature search, correct, when you evaluate a new exposure?

A Yes.

Q You go to files you've assembled and have literature on a particular exposure and you pull all that

out?

A Yes.

Q And I'm assuming you take the box or the stack and put it on your desk or your conference room and have the literature in front of you, is that one of the steps?

A Yes.

Q Is there a method you can show me, a piece of paper or a ranking sheet or that you can describe for me whereby you separate the wheat from the chaff when you have your epi studies in front of you?

A There are a lot of different things you look at, the selection of the population, and one of the powerful things with cancer studies is you have proper latency.

Do you have a number of people who have long enough time from the onset of exposure to identify whether there is an excess cancer risk or have you loaded it all with people who have less than 15 years from the onset of exposure, in which case you look at the cancer and you make a big chunk of your population people that have not got proper latency and try to include them in your study and you have a bad study.

So, you know, it depends. If you're looking for the prevalence of asthma from toluene de iso cyanate, you don't care about latency because it's a disease that

occurs after a short time after the exposure.

So you want dose and a decent population size and proper statistical power and a well-matched control group. Then there's a lot of other sub questions you want to ask, depending what end point you're looking at.

Q What you've given me is a list of probably what are great considerations that you would apply as you look at individual studies and determine its quality.

But my question is I think more simple. Is there a method you have whereby you rank studies -- an A pile, B pile and C pile?

A I don't usually actually rank them. When I read them and analyze them, look at the data, I put it into my framework and some papers add very little information for various reasons and others add a lot more information for various reasons, and there is a spectrum but I don't necessarily go in and grade them. As I said, it depends what questions you're trying to derive from these different studies.

Q Other than to tell me you read the studies and mentally order them along the spectrum that you just described, is there any other way you can describe for me the method for evaluating epidemiologic studies to come to a causation?

A Other than what I've said, no.

MR. LUNDY: Can we take a break?

MR. HOPP: Couple more and we can.

Q Do you think that that method you just described for me is reproducible?

A Sure.

Q Do you think that any other epidemiologists with similar training and qualifications to you would reach the same conclusions of applying your spectrum method?

A I think so, yes.

MR. HOPP: Now we'll break.

(Lunch break.)

BY MR. HOPP:

Q Dr. Dahlgren, we're back on the record and we'll focus on the 12 plaintiffs, and feel free to use your notes, and I have your medical summaries on the 12 plaintiffs and we'll go through those in some detail, most likely tomorrow, but let's go through them one at a time and tell me generally your opinions.

Do you have opinions regarding each of the 12 plaintiffs in this case?

A Yes.

Q As we covered earlier this morning, opinions relating to causation and increased risk of disease?

A Yes.

Q I'd like to start with Patricia McNeal.
A I've got the report in front of me.
Q And for the record, you're looking at your laptop computer, and Mr. Lundy's office produced to us three disks containing your files in this case.
Have you turned over to Mr. Lundy's office all of the material reports and various data you compiled on these 12 plaintiffs?
A Yes.
Q And what you're looking at there, and we may confirm this tomorrow most likely, is a medical report you created on Ms. McNeal or a summary of some type?
A Yes.
Q That's one of the documents you turned over in this case?
A Yes.
Q Tell us generally your opinions with respect to Patricia McNeal.
As you're looking at your report, can you tell us the date of the report you're looking at?
A The date of examinations is October 24, 2004 and the -- I don't have a specific date when the report was prepared.
But in terms of my opinion about her, I believe she was exposed significantly to PAHs, dioxins and

pentachlorophenol while living at Carver Circle, and has lived there for many years -- it's listed here she lived there for 26 years. She didn't always live on Carver Circle but in that vicinity.
That she smelled odor when she lived at Carver Circle in particular, and this caused her to have a reduced sense of smell, which meant then after a while she didn't smell it anymore, and it didn't mean it was not there but that her smell became impaired and I think was recently documented by Dr. O'Jile, who did do a test on her smelling capacity and, according this smell test, she has anosmia and can't smell at all. And she complained about that, that as a consequence of being in the neighborhood, she lost her sense of smell.
They used a local well that she believed was contaminated with -- there was a well they had on Carver Circle, and they used to take water from that well for many years, and about five years prior to my examination they switched to city water, but before that they -- she believes that well was contaminated, although I don't have any measurements that I have seen for that opinion.
Her biggest health problem based on her history was severe asthma and she also has very severe hypertension. Those are her two biggest health problems.
Q Hypertension?

A Correct.
Q Any other --
A She has some other health problems which we can go through. Let me go to the summation page --
Q What I'd like to know for this and for each of the other plaintiffs, and we'll go through it one by one, is which of their health problems do you believe was a result of a chemically-induced injury. If she has health problems that are unrelated, let's not spend time on that.
A Not discuss them?
Q Not right now.
A Okey-dokey. Related to the exposure, she has neurologic complaints, and it's not probably entirely due to her chemical exposure and probably a contribution from her hypertension but she has headache, dizziness, lightheadedness, loss of balance, loss of consciousness, fatigue, somnolence, insomnia, irritability, lack of concentration, recent and long-term memory loss, instability of mood, decreased libido and driving confusion, and I believe those neurologic complaints have been aggravated by living where she did with the exposures to the chemicals we've been talking about.
As I stated, she has significant asthma for which she's on various medications and her symptoms from

her chest condition include chronic productive cough, sometimes tinged with blood, chest tightness, pain in the chest, shortness of breath, dry mouth, nose and throat irritation and sore throat and sore nose and runny nose, eye irritation, reduced sense of smell -- which is probably a combination of neurologic and her respiratory effects -- sinusitis.
She also has some autoimmune symptoms, which have been aggravated by her exposure. She has sores in her mouth, low blood count, anemia, low white blood cell count, low platelet count, rash on her cheek and some skin rash when her skin breaks out when out in the sun, and this is photosensitivity, very characteristic of creosote poisoning. Additional skin problems are excessive dryness and itching and noticed changes in her fingernails. All these are characteristic of creosote exposure.
She has had two different cancers. One is skin cancer diagnosed in 1994 and a cervical cancer, although I need to -- that's in her medical records. She didn't fill it out on her questionnaire and probably forgot about it. She was diagnosed with carcinoma in situ of the cervix, and she reported that as a skin cancer but it's really a squamous cell carcinoma in situ of the cervix, and that's in 1994, so it was only one cancer.

I think those are the conditions that have been caused or aggravated by her exposures.

Q You said caused or aggravated. Have you made any specific opinions about which of her conditions were caused by exposure to PAHs, dioxins and penta and which were merely aggravated by those exposures.

A Well, her hypertension was contributing to her neurologic symptoms and I didn't opine that her hypertension was related to the exposure. It might have been but I'm not offering that opinion at this time. So that the contribution from her hypertension to her neurologic symptoms is significant and her blood pressure is quite high.

Q I have to interrupt so we can complete that thought. She has hypertension since before she was ever lived in the neighborhood; is that right?

A Yes. She had hypertension with her first pregnancy when she was 16 years old, and that's clearly predated her moving to that location.

Q And it's your opinion that preexisting hypertension has caused some of her neurological injuries?

A That's what I said, yes.

Q And I want to make sure I understand. Is it also your opinion that her exposure to PAHs, dioxins and

penta have also contributed to those neurologic conditions?

A Yes; the exposure to the chemicals we discussed, the penta, the PAHs and dioxins have contributed to the neurologic problems and made them worse.

Q Can you tell me what neurologic problems Ms. McNeal would have had had she not been exposed to penta, creosote or dioxins in the Carver Circle?

A I have no way to meaningfully apportion those factors.

Q So you can't tell me whether her neurological conditions were aggravated 10 percent, 5 percent or 90 percent by exposure; is that correct?

A Well, I think I can say that both are significant contributing factors to her current neurological picture, and I don't know any meaningful way to separate the two.

Q So but for the exposure to creosote, penta and dioxin, Ms. McNeal would have hypertension and resulting neurological problems; is that correct?

A I can't say that. I can say that patients with hypertension, particularly if severe, do develop neurologic problems but it's incorrect to say that she would have had these neurologic symptoms from her

hypertension alone. All we can say is that she has these health problems, and they are the result of the combination of exposures that we see here.

Q Are there any particular neurological problems you can separate out, that is ones she would have had anyway and ones she now has, as a result of the exposure, that she would not have had had she not lived in the neighborhood?

A It's the same question basically. There's no way to say that she would have had this problem had she just had hypertension and lived somewhere else. It's sort of -- you can't talk about such a hypothetical with any basis. We have to talk about what we have in front of us, which is a patient with these health problems, and we know that hypertension is a factor in causing these in some people and not all. Some patients with hypertension don't have any of these symptoms.

If my understanding of this whole problem is correct, she is more likely to have them from her exposure because in her neighborhood a lot of people have these symptoms, and I've examined hundreds of patients with hypertension and many of them or most of them are asymptomatic, and I would say that the most likely situation here is that she has these symptoms from her exposure rather than hypertension, but I can't really be

dogmatic about that.

Q Ms. McNeal has severe hypertension; is that correct?

A She does have severe hypertension.

Q And in someone with severe hypertension, would it surprise you to see some neurological symptoms?

A As I stated, one of the known side effects of hypertension and severe hypertension. But, again, there are patients that come in with hypertension, severe, where they don't have it, and you can't make the statement that she would have had it for sure absent -- it would be incorrect to say for sure that she would have had these things just looking from what her blood pressure was when I saw her. I'm pretty sure she was quite high but I'm not finding the blood pressure here.

Q Let's phrase it a different way or ask a different way.

Without asking you to say yes or no, is it more likely that a patient with severe hypertension is going to have neurological symptoms, even if it doesn't always happen?

A Yes, depending on the severity of the blood pressure and the response to medication, she would be more likely if she had severe uncontrolled hypertension to have neurologic symptoms rather than not.